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Heart Failure and Cardiomyopathies

POST-DIURETIC COMPENSATORY SODIUM RETENTION IS NOT A PREVALENT FINDING IN PATIENTS WITH HEART FAILURE

Poster Contributions

Poster Hall B1

Saturday, March 14, 2015, 3:45 p.m.-4:30 p.m.

Session Title: Advances in Heart Failure Therapies: From Diuretics to VADs and Transplant

Abstract Category: 14. Heart Failure and Cardiomyopathies: Clinical

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Background: One of the core principals in our understanding of diuretic resistance is that, during the post-diuretic period, the kidney will maximally reabsorb sodium in an effort to compensate for prior diuretic-induced sodium loss. Derived from studies of normal and hypertensive patients, this concept forms the basis upon which continuous loop diuretic (LD) infusion was hypothesized to be superior to bolus therapy. However, recent trials in heart failure (HF) patients have failed to demonstrate meaningful superiority of diuretic infusion. Our objective was to determine if significant post-diuretic compensatory sodium reabsorption was in fact a common finding in HF.

Methods: Urine sodium concentrations were obtained in a diverse cohort of HF patients at least 8 hours following the last dose of LD (n=99). The cohort included 29 decompensated patients receiving IV diuretics and 70 stable patients receiving oral diuretics.

Results: The median dose of LD was 120 mg (70 to 180 mg) of furosemide equivalents. On average, pre-diuretic urinary sodium levels were not consistent with severe post-diuretic sodium hyper-avidity (47 ± 34 mmol/L). There was significant inter-individual variability in the urine sodium concentrations (range 2-193 mmol/L). Notably, 8.1% of patients had urine sodium concentrations >100 mmol/L and 26.3% >60 mmol/L. Only 18.2% had a urine sodium <20 mmol/L and 6.1% <10 mmol/L. The mean urinary sodium levels were not different between hospitalized patients (48 ± 36 mmol/L) and outpatients (47 ± 32 mmol/L, $p=0.58$). There were no differences between patients receiving a loop diuretic dose above or below the median ($p=0.91$).

Conclusion: Severe post-LD compensatory sodium reabsorption is not a consistent finding in patients with HF. A high urine sodium concentration was as common as a low value. This was true in both stable outpatients and decompensated inpatients. These observations may help to explain the lack of benefit from continuous LD infusion in unselected HF populations. Future research is necessary to determine whether continuous infusion could enhance diuresis in the subset of patients with significant post-dose compensatory sodium reabsorption.